THE RELATION OF TYPHOID FEVER TO TUBERCULOSIS.

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It is well known that typhoid fever and acute tuberculosis may be rarely associated, and form, so to speak, a composite clinical picture. More commonly, typhoid fever develops in the course of chronic tuberculosis. When acute miliary tuberculosis and typhoid are concurrent, the former disease is, as a rule, secondary to the latter, although it must be recollected that an old tuberculous focus usually co-exists.

In other words, these are cases in which persons with either an active chronic tuberculosis or a latent encapsulated focus contract typhoid fever. It may be assumed that the reaction of the tissues is incomplete; that the resisting powers are defeated and the bacilli liberated under the influence of the typhoid infection. It is also clear that forms of acute tuberculosis are not likely to supervene when there is present a thick, dense limiting wall as in old cases that are practically cured.

This paper deals principally with typhoid fever complicated with acute miliary tuberculosis and acute pneumonic phthisis. It is important to bear in mind that the typhoid process is responsible, in the manner explained above, for the rapid dissemination of the tubercle bacilli or the conversion of chronic into acute tuberculosis.

It must be admitted that rapid infection of the lungs with tubercle bacilli from without may also occur in the course of typhoid fever and acute pneumonic phthisis result as a complication, the primary infection serving as a predisposing condition. Indeed, in at least 4 of the six cases of combined typhoid and acute tuberculosis reported below, the latter affection assumed the pneumonic type, and some of these at all events may have been due to this mode of infection. Moreover, it must be recollected that when typhoid fever arises in subjects of arrested pulmonary tuberculosis and is complicated with bronchopneumonia (as was true of Case II. of my series) the inflammatory changes occasioned by this complication may break down the protecting wall and allow of the distribution of the tubercle bacilli throughout the lungs as well as to other and more remote regions by means of blood and lymphatic metastasis.

It does not appear that a chronic tuberculous process predisposes markedly to typhoid fever, judging from the comparative infrequency of enteric fever in the course of that common disease. Thus out of a totality of 249 autopsies in cases of typhoid fever, only 19 (7.6 per cent.) showed the presence of old tuberculous lesions. Per contra, the former widespread opinion that typhoid fever predisposes to tuberculosis receives no support from my observations, as will be shown hereafter.

Eshner[1] has reported a case of triple infection—pulmonary tuberculosis with intercurrent typhoid fever complicated by pneumonia—and, bearing upon the subject of the occurrence of typhoid fever in the course of tuberculosis, he makes the following statistical quotations: "Among 2000 fatal cases of typhoid fever examined after death at Münich, Hölscher noted 108 (5.4 per cent.) of tuberculosis in various situations, and he quotes Gruber as having noted, among 710 fatal cases of typhoid fever, 22 (3.3 per cent.) complicated by old tuberculosis of the lungs. Bettke, among 1420 cases of typhoid fever at Basle, noted 23 (1.6 per cent.) in tuberculous subjects; while Dopfer noted tuberculosis in 46 (5 per cent.) among 927 cases of typhoid fever."

No allusion is made to the association of acute forms of tuberculosis with typhoid fever in these statistical representations. M. Forget[2] affirms that persons suffering from pulmonary tuberculosis are rarely attacked by typhoid fever, and regards the former as preservative against the latter.

The question of the diagnosis of these combined affections is of the utmost importance from the standpoint of prognosis, the outlook in pure typhoid being generally hopeful. I have also endeavored to determine the incidence of the hybrid affection—typhoid fever and acute tuberculosis—which, as will be shown hereafter, is fortunately extremely rare. The writer has recently met 2 cases in his hospital practice in which these two leading affections appeared to be concurrent. The post-mortem records of the Philadelphia and Episcopal Hospitals for the last ten years have been carefully examined with reference to the question of the relation of typhoid fever to tuberculosis, and the results have been tabulated as follows:[3]

	Total number of cases showing typhoid lesions.	Number of cases showing both typhoid and tuberculous lesions.	Number showing		Variety of acute tuberculosis.
			typhoid and chronic tuberculosis.	typhoid and acute tuberculosis.	
Philadelphia Hospital.	90	11	10	1	Acute miliary tuberculosis.
Johns Hopkins Hospital.	80	4	3	1	Acute pneumonic phthisis.
Episcopal Hospital.	79	8	6	2	Acute pneumonic phthisis and miliary of lungs and pleuræ.
Summary	249	23	19	4	

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[1] Philadelphia Medical Journal, March 25, 1899.

Quoted by Murchison, The Continued Fevers of Great Britain, third edition, London, 1884.

[3] The writer desires to make grateful acknowledgment to Dr. Wm. Egbert Robertson and Dr. Ralph H. Spangler for kind aid in collecting statistical data.

Cases illustrative of combined typhoid and acute pneumonic phthisis and acute miliary tuberculosis.

CASE I.—L. B., aged twenty-five years, colored, unmarried, and by occupation a domestic; no history of the use of alcohol, but has had syphilis and one miscarriage; menstruation was regular as a rule.

Family History. Father deceased of unknown cause; mother of pneumonia; patient statedth at she was sick all of last winter; she died in spring; patient had never had any brothers or sisters.

Personal History. Whooping-cough, measles, and mumps as a child; at eighteen years had an attack of rheumatism lasting two weeks and making good recovery. Colds and "coughs" have been common during second decade of life; also sore throat, and much headache.

Present Illness. August 23d went to Cape May and contracted a "cold," which became persistently worse, and she was obliged to betake herself to bed. On September 8th patient was admitted to the female wards of the Medico-Chirurgical Hospital with a cough, pain in her left axillary region, fever, etc.

Physical Examination. Young colored woman, medium size, with poor development and slightly emaciated. There are visible a considerable number of punched scars on the anterior surface of leg. Knee-jerks normal; sensation normal; eyes, liver, and heart also normal. Spleen somewhat enlarged; consolidation of lower lobe of left lung, with a small cavity in left upper lobe.

September 18, 1903, 5 P.M. On account of the splenic enlargement the blood was tested for the Widal reaction, and it responded positively. A sero reaction was also obtained on September 20th and again on September 30th; the diazo reaction was present; considerable enlargement of spleen. Cardiac impulse weak and diffuse pulsation felt over region of left auricle. Slight increase of dulness over same region, but no murmur detectable. Patient much distressed; feels certain that death is at hand. Has at no time been delirious, but symptoms of motor irritation were noted as tremor of upper lips and hands.

Blood Examination. Hæmoglobin, 75 per cent.; reds, 4,680,000; whites, 9400.

Urine Examination. Granular casts; albumin, a trace; specific gravity, 1012.

21*st*. *Sputum Examination.* Tubercle bacilli present; also elastic tissue.

There would appear at first sight to be no doubt as to the coexistence of typhoid fever and acute pneumonic phthisis in this case. The earlier examinations of the patient after admission into the hospital all seemed to indicate tuberculosis; the sole feature that directed attention to the typhoid infection was some considerable splenic enlargement—a more marked increase in size than I had ever seen in pure acute pneumonic phthisis. This fact suggested the making of a blood examination for the Widal reaction and led to the discovery of the associated disease—typhoid fever. Other symptoms of enteric fever, however, developed subsequently.

Case II.—J. S., aged twenty-eight years, a Polander, single, laborer, was admitted to the Medico-Chirurgical Hospital October 29, 1903. The family history proved negative, and the same is true of his previous personal history. Patient was well developed and well nourished on admission; he was rational, though considerably prostrated and temperature 102° F.; spleen enlarged and two rose-colored spots visible. In addition, there are noted bluish spots, the "taches bleuatres" of the French writers, ranging from one-third to one-half inch in diameter and scattered over the entire body; they do not disappear upon pressure. A noticeable enlargement of the postcervical or epitrochlear glands can be detected; the penis shows an old linear scar beneath the frenum. There is slight rigidity of neck, but no tenderness along spine, scalp or head; the conjunctivæ are injected, the pupils slightly dilated. The gums are inflamed at the teeth, and bleed upon the slightest touch, and the pharynx is the seat of a decided catarrhal irritation. Numerous moist rales are heard over both lungs, more especially at right base, where the breath sounds are also bronchovesicular, showing lobular infiltration. Heart normal, although pulmonic sound is slightly accentuated. The spleen is enlarged; the abdomen not tender, but somewhat tympanitic with gurgling to pressure in right iliac region.

November 4th. General symptoms increased in severity; eruption characteristic and abundant; had rigor previous night, and later slight intestinal hemorrhage. The physical signs indicate resolution in right lower lobe of the lung. A bandage applied to right elbow after taking a blood culture produced ecchymosis of right forearm. Has marked hiccough, and is delirious.

12th. The physical signs indicate an area of consolidation in left lung near the base, and also one on right side at angle of scapula (bronchopneumonia). The sputum is tenacious, and decidedly hemorrhagic, and has been so for twenty-four hours; it shows no tubercle bacilli but streptococci and staphylococci. The urine analysis revealed the findings of an acute hemorrhagic nephritis.

15th. Small mass is noted in lower right quadrant of abdomen, below (?) the seat of the appendix. It is soft and presents fluctuation, and is slightly painful to touch.

18th. Patient's general condition has improved, although a large hemorrhage occurred last evening, accompanied by the usual drop in temperature. The palpable mass in abdomen is still present without change; the surgeon refuses to operate on account of extensive bronchopneumonia. The sputum is viscid, and tinged with specks of red blood. Signs of resolution slowly progressing in right lung.

December 10th. Since former note, numerous examinations of the sputum were made, with the same result as noted above. The Widal reaction was noted a few days after admission, and on several occasions during the course of the illness; the diazo reaction was also present.

11th. The physical signs indicated the presence of a small cavity in the left interscapular space. It was thought that an abscess had developed, but the sputum examination on the following day showed the presence of numerous tubercle bacilli for the first time, but subsequently they were constantly present and the temperature became hectic in type. The conclusion was now inevitable that tuberculosis had probably been associated with typhoid fever, and only declared itself during early convalescence.

The next case herein reported occurred in the Philadelphia Hospital, and is taken from the post-mortem and clinical records of that institution; it is the only instance of combined diseases under consideration out of 90 autopsies on cases of typhoid fever and on all cases of acute tuberculosis covering a period of ten years.

CASE III.—G. K., aged twenty-one years, white, single, laborer, admitted April 18, 1899. The history sheet was lost, but a few ward notes were found; the temperature averaged from 102° to 103.2° F., pulse 72, later frequent; respirations about 26; the spleen was enlarged, the abdomen was tympanitic and diarrhœa was present; sero reaction present; tubercle bacilli absent at first, later found; no mention of rose spots. Condition grew progressively worse, and patient died May 21, 1899.

The autopsy showed intestinal perforation with adhesions to the omentum. Typhoid ulcers in large intestine cicatrizing; also ulcers about the valve with swollen solitary follicles. Peyer's patches in stage of infiltration and ulcers very numerous.

The left lung is almost entirely free from pigmentation; the anterior portion of lung normal, except a few tubercles scattered through the apex; posteriorly, there are patches of pneumonia, involving about one-half the depth of the entire posterior portion of lungs. Right lung: upper lobe, normal; middle lobe is tubercular throughout, being consolidated to a considerable extent with many tubercles; lower lobe shows hypostatic pneumonia almost throughout.

The post-mortem records of the Episcopal Hospital furnish two instances of combined typhoid fever and acute tuberculosis, as follows:[1]

CASE IV.—J. McI., aged eighteen years, single, was admitted to the medical wards of the Episcopal Hospital August 30, 1898, and died September 12, of the same year.

The clinical diagnosis was typhoid fever with perforation. The autopsy, however, showed acute miliary tuberculosis in addition to the typhoid lesions with perforation. Both lungs were studded throughout with miliary tubercles, but there was no consolidation. The spleen was enlarged, and the small intestines presented typhoid lesions in the usual seat associated with marked hyperæmia. Perforation had taken place in the ileum, around which there was an adhesive, localized peritonitis. Many of the lesions were about to show necrosis.

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[1] The writer regrets that no clinical notes of this case are available.

CASE V.—On admission, the diagnosis of typhoid fever accompanied the patient, but the clinical diagnosis during the stay in the hospital was pulmonary tuberculosis and tubercular bacilli were found in the sputum. The case proved fatal, and loculated, serofibrinous, and hemorrhagic pleurisy was noted at autopsy; also caseous deposits and miliary tubercles throughout lungs and pleura, while the intestines presented typical typhoid ulcers. In this instance of combined typhoid fever and acute tuberculosis both affections had been, although at different times, diagnosticated during life, but their association was not recognized.

CASE VI.—Osler has informed me that out of a total of 80 autopsies of cases of typhoid fever at the Johns Hopkins Hospital, one showed acute tuberculosis starting in as an acute pneumonic condition.

It is evident from the foregoing statistical facts including the reports of the 5 cases, that combined typhoid infection and acute tuberculosis, as stated above, is rare. Thus acute tuberculosis was associated in 1.6 per cent., and chronic tuberculosis in 7.6 per cent. of the cases of typhoid that came to autopsy. I have not included case histories (except Nos. 1 and 2), unsupported by post-mortem proof, for the reason that the chances of error in diagnosis are exceedingly great, as will appear hereafter.

It is said that when tuberculosis and typhoid fever are associated together in the same case, the former may dominate the scene and cause the Widal reaction either to disappear or to fail to appear. For this reason, certain cases of the combined disorders may be readily overlooked.

E. Kraus[1] found in 1 case that a complicating pneumonia caused the sero reaction to disappear just one day prior to the death of the patient. The post-mortem revealed the lesions of typhoid and lobar pneumonia in combination. It would appear from this fact that the serum of some other disease like pneumonia and acute tuberculosis may prevent the reaction of the typhoid bacilli. It is possible that the sero reaction of typhoid fever is sometimes also delayed on account of interfering complications, or again, it may be temporarily absent in the course of a given case for the same reason. A careful study of the cases reported in the literature will reveal much evidence to support this view. Thus cases complicated with appendicitis at times, as I know from personal observation, do not show the Widal reaction until the complication is removed by operation.

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[1] Zeit. f. Heilk., Bd. xxi., H. 5.

Conversely, F. Lobiesen[1] examined the blood of 151 patients who did not have typhoid fever, but were admitted into the hospital with that diagnosis or suspected enteric fever. Of these 4 gave a positive reaction at 1 to 25, and 3 died, showing respectively acute miliary tuberculosis, retroperitoneal abscess and calculous pyelitis with tuberculous meningitis. He, with Fiocca,[2] concludes that a positive diagnosis can be made when the reaction occurs with a dilution as great as 1:50. Now, in Case I. of my series the blood examinations were made by the Bacteriological Department of the Bureau of Health, where the higher dilutions (1:40 to 1:50) are employed. It was deeply regretted that no autopsy was allowed, and yet, as stated above, there can be little room for doubting the coincidence of typhoid fever and acute pneumonic phthisis in view of the three positive sero reactions at intervals of several days. There was absolutely no history of previous typhoid infection.

If the Widal reaction can be relied upon to differentiate typhoid fever from acute tuberculosis with absolute certainty, then these two diseases were associated in this instance. In 2 of the cases reported below, however, in which the sero reaction occurred, the necropsy failed to show any typhoid lesions.

A number of recent observers have found that icteric serum was possessed of a certain amount of agglutinizing power. The subject has just been carefully reviewed by E. Libman.[3] He quotes Koehler, who found in 8 cases of icteric diseases of the liver an agglutination reaction in 6. The highest dilution in which he obtained the reaction was 1:40; very rarely 1:50, and once it was only 1:10. Koehler experimented with the bile of rabbits which had slight agglutinizing power; also with that of dogs, and this manifested a decided agglutinizing potency (even up to 1:160).

Libman[4] made observations upon ten specimens of human bile, either normal or from cases of typhoid fever, chronic pancreatitis or cholelithiasis, and in no instance could he get a positive Widal reaction. Sailer[5] reports 3 cases of intense jaundice, all of which failed to give the sero reaction. The time has arrived when a *slight reaction* in a dilution of 1:50 is not to be regarded as a positive Widal reaction.

It is admittedly difficult to interpret the clinical phenomena of the combined diseases during life. All of the cases that came to autopsy showed that the clinical diagnosis was either incomplete or erroneous. In Cases I. and II. (see *ante*) acute pneumonic phthisis was associated with typhoid fever, and in the former a lobar type of consolidation occurred, while in the latter it was a bronchopneumonic type.

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[1] Zeit. f. klin. Med., D. V. L. 111, Heft 1 und 11.

[2] Il Policlinico, November 1, 1900.

[3] Medical News, January 30, 1904.

[4] Loc. cit.

[5] Transactions of the Philadelphia Pathological Society, 1902, p. 253.

The question of the effect of acute tuberculosis upon the course of typhoid fever is one of considerable interest. It was observed in Case I. that the typhoid fever was apparently uninfluenced by the tuberculous process. Indeed, it may be said that the symptoms of typhoid were distinctly in abeyance. In Case II. the clinical features of typhoid were intense and the course prolonged, and yet this disease was a thing of the past before the tuberculous process reached the stage of cavity formation, and tubercle bacilli found in the sputum.

The temperature-curve in the combined cases resembled closely that of typical typhoid—at all events until the typhoid process had spent its main force. On the other hand, the pulse partook during the fastigium more of the character of that seen in acute tuberculosis than of typhoid fever, being rapid and feeble.

The diagnosis of the combined affections may be assisted by remembering that the characteristic typhoid symptoms usually manifest themselves before the diagnostic evidences of acute tuberculosis. This is particularly true of the cases in which the pneumonic type of a tuberculous process is associated with typhoid. Here the diagnosis cannot be completed, as a rule, until tubercle bacilli are discovered in the sputum usually after the lapse of weeks. In my cases no tubercle bacilli were detected until the physical signs denoting apical cavity formation were noted. In this connection, attention should again be called to the fact that in the majority of the cases the associated tuberculosis assumes the form of acute pneumonic phthisis, according to the results of my collective investigations. Certain forms of acute tuberculosis were commonly mistaken for typhoid fever, such as acute miliary tuberculosis and the acute pulmonary varieties, as the reports of the following cases will illustrate:

In November, 1901, I had under my care a patient in the Medico-Chirurgical Hospital suffering from typhoid fever, and who gave a strong family history of pulmonary tuberculosis. The case began with pleuritis, accompanied with moderate effusion. The diagnosis of tuberculous pleurisy was provisionally made, but after the lapse of ten days true typhoid symptoms supervened, while the effusion was slowly and gradually absorbed. Recovery ensued.

An interesting and instructive case is recorded by Osler,[1] in which for nearly two weeks tuberculous pneumonia was suspected on account of apparently clear physical signs and the family history. Later, the true typhoid symptoms appeared and became well marked, although the Widal reaction was not present until the end of the third week.

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[1] Studies in Typhoid Fever, series 3, p. 464.

The following two case histories bearing on this point have been taken from the clinical and post-mortem records of the Philadelphia Hospital:

CASE I.—S. R., aged twenty-two years, Polander, occupation laborer, was admitted to the medical wards of the Philadelphia Hospital April 26, 1902; a chill one week before, followed by dyspnœa, cough, bloody expectoration, intestinal disturbance, and mental dulness. No family history obtainable. The physical examination showed coated tongue, dicrotic pulse, bronchovesicular breathing, and impairment of resonance over lower lobes. The abdomen was distended and tympanitic; spleen enlarged; few suspicious rose spots observed; temperature ranging from 102° to 104° F.; pulse 66 to 100; no positive Widal reaction; diagnosis of pulmonary tuberculosis was made during life.

The autopsy revealed typical typhoid ulcers, acute splenic tumor, parenchymatous degeneration of the liver and kidneys, and congestion of the lungs. On section, the lung texture showed slight increase of consistency, but crepitation was everywhere present. Typhoid ulcers were found in the ileum, and several large ulcers in the cæcum.

CASE II.—C. S., aged thirty-six years, a patient in the medical wards of the Philadelphia Hospital, died February 6, 1902. The history of the case was as follows: Ill three weeks with chill, cough, slight expectoration, occasional vomiting, some abdominal pain, diarrhœa, with bloody stools twenty-four hours after admission; temperature 104° F. Patient passed into the typhoid state, but no rose spots and no splenic enlargement was detectable. On percussion the bases of the lungs showed dulness, and on auscultation crepitant rales were heard over both lungs. A trace of albumin was found in the urine; the sputum showed no tubercle bacilli; Widal reaction not mentioned. The diagnosis during life was acute tuberculosis.

The post-mortem revealed typhoid ulceration of the intestines, acute splenic tumor, hyperplasia of mesenteric and other lymph glands, œdema of lungs and apparent parenchymatous degeneration of the kidneys. The lungs, it may be mentioned, showed increased consistency, and on section a dark and blood-stained fluid exuded upon pressure; no evidence of consolidation, and bronchial lymph glands negative.

Typhoid fever may be mistaken for tuberculosis, as shown by the following illustrative cases:

CASE I.—J. P., aged twenty-five years, colored, laborer, admitted April 23, 1899; family history negative; had malaria one and pneumonia three years previously. Present illness began two weeks ago with typical prodromes of typhoid; a few days later was seized with pain in left side, and cough with slight expectoration. The above symptoms lasted until admission, after which he had two or three stools daily; also high temperature of irregular type; pulse small, frequent, and little tension; slight albuminuria; a few dry rales at end of inspiration. The Widal reaction was present and a few tubercle bacilli were found in sputum. The clinical diagnosis had been typhoid fever and tuberculosis.

The patient died twenty-eight days after admission, and the necropsy showed only tuberculous lesions, the intestines being normal (not injected even); tuberculosis of the mesenteric, retroperitoneal and periaortic lymph glands, and a small nodule in the liver. Miliary tuberculosis in lungs and spleen. Pathological diagnosis was chronic followed by miliary tuberculosis.

It will be noted that the sero reaction was found in this case without the presence of any discoverable typhoid lesions. It must be recollected, however, that cases of typhoid septicæmia occur in which there may be an utter absence of characteristic intestinal ulcers. It is with considerable unreadiness that I place on record this case and the following, both of which tend to disprove a dictum which has been generally accepted, while searching for data to show the interrelation of these affections.

Case II.—H. M., aged twenty-four years, occupation brakeman, was admitted to the male medical wards of the Philadelphia Hospital August 2, 1903. Family history negative; the patient had always been healthy except that he contracted syphilis two years previously. He was a confirmed alcoholic and an excessive smoker. On admission, was thought to be intoxicated, and taken to the "drunk ward," but the symptoms and appearance did not portray drunkenness; he was semiconscious, although in muttering delirium. It was learned that he had been indisposed for two weeks with anorexia, headache, increasing prostration. The spleen was slightly enlarged; the diazo reaction negative, Widal positive, and a few suspicious spots were noted on the back; the physical signs seemed to indicate lobar consolidation, and the respirations were as high as 40 per minute. A clinical diagnosis of typhoid fever complicated with pneumonia was made.

The patient died August 12, 1903. An autopsy revealed encysted pleural effusion and miliary tuberculosis; the intestines showed no macroscopic changes.

Case III.—C. H., aged twenty-five years, admitted to the Philadelphia Hospital March 20, 1900. Nothing could be gleaned regarding either the family or personal history. Said to have been ill two weeks, beginning with headache, malaise, diarrhœa, and high fever, but no cough. Physical examination shows tenderness, and gurgling in right iliac fossa, splenic region tender, but spleen not palpable. On sixth day after admission he became delirious, spat up a couple mouthfuls of blood; next day passed a quantity of dark blood with stool. The urine contained both albumin and tube casts; the temperature ranged from 101° to 102° F. for six days, then from 99° to 100° F. until death, which occurred March 28, 1900.

The clinical diagnosis was typhoid fever. Post-mortem examination revealed acute tuberculosis; left lung rather firm, but crepitant throughout; no tuberculous cavity or nodes. Right lung at apex showed marked tuberculous change; spleen normal in size; the intestines showed numerous tuberculous ulcers.

CASE IV.—C. L. G., female, aged thirty years, died April 9, 1899, at the Episcopal Hospital (no bedside notes obtainable). A clinical diagnosis of typhoid fever was made, but the pathological findings showed the following: Lungs riddled with miliary tubercles, although no source of infection was discoverable; neither caseation nor cavities were observed. Bronchial glands slightly enlarged, but not broken down; the spleen weighed 6½ ounces, was very soft and friable, with some miliary tubercles scattered through its surface. The intestines were perfectly normal; the peritoneal surface of the bladder and uterus was studded with a few tubercles.

CASE V.—Patient was admitted to the medical wards of the Episcopal Hospital believed to be suffering from typhoid fever. The disease ran an acute course and proved fatal. At autopsy, miliary tuberculosis following chronic pulmonary tuberculosis, with cavity formation in lungs and also intestinal tuberculosis, was found.

CASE VI.—On admission, this case was diagnosticated as typhoid fever, but had typhoid six years previously. The Widal reaction was present; leukocytes, 10,400. Blood cultures were made, but did not show typhoid bacilli, and lumbar puncture failed to elicit fluid. The necropsy showed acute meningitis, especially at the base, associated with pleural adhesions, cavities in both lungs, and tuberculous ulcers in the intestines.

The question here arises, Was the Widal reaction in this case due to the attack of typhoid fever which had occurred six years previously?

Osler[1] has reported 3 interesting cases of the sort in which at the outset typhoid fever was strongly suspected, although the positive signs of this disease were absent, and the certain features of pulmonary tuberculosis finally supervened. In 1 of the cases, as is usual, rapid progress was made during lactation and the fever was mistaken for typhoid.

While it is important to recognize these two leading affections when combined, remembering the extreme rarity of the association, it becomes a matter of greater clinical importance to distinguish between typhoid fever and the various forms of acute tuberculosis, since they are so commonly confused with one another. Acute miliary tuberculosis may simulate typhoid fever so closely as to offer the greatest clinical difficulty. The absence of the Widal reaction, of the characteristic eruption and of the comparatively slow pulse in miliary tuberculosis, are to be especially noted. On the other hand, in cases in which strong suspicions of the existence of acute miliary tuberculosis are entertained, a careful search for tubercles in the choroid, and tubercle bacilli in the blood and sputum (if there be any), is to be carried forward. C. Loeb[1] has recently emphasized the value of ophthalmic examinations in the differential diagnosis between typhoid fever and acute miliary tuberculosis. The personal history is often most helpful. In doubtful cases an attempt should be made to cultivate the typhoid bacilli from any suspicious eruption that may be present. There are not a few cases in which a positive diagnosis, however, must be withheld, for lack of discriminating features.

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[1] Loc. cit.

DISTRIBUTION OF THE ULCERS IN TYPHOID FEVER.

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THE occurrence of two cases of typhoid fever with an unusual distribution of the ulcers suggested the desirability of a general investigation of the distribution of ulcers in this disease. The two cases are presented in detail, and are followed by an analysis of a series of 89 cases, and by a review and discussion of the literature on the subject.

The first case showed ulcers in the œsophagus and colon, and in no other part of the alimentary tract:

CASE I.—This series 33; Rush Medical series 945. A. B., male, aged twenty-four years; died January 31, 1901.

Clinical Diagnosis. Typhoid fever; tonsillitis; pharyngitis; laryngitis; pleuritis; probable pneumonia.

Anatomical Diagnosis (Dr. H. G. Wells). Typhoid fever (colotyphoid); ulcerative colitis; follicular enteritis; swelling of mesenteric lymph glands and spleen; follicular pharyngitis; œsophageal ulcers; acute serofibrinous pleuritis; bronchopneumonia; fibrous perisplenitis and perihepatitis; healed pulmonary tuberculosis.

Tongue. Lymphoid follicles prominent.

Pharynx and Epiglottis. Covered by mucopus.

Œsophagus. Covered by thin mucopus. There are prominent follicular swellings. At a point 1 cm. below the cricoid cartilage is

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[1] Archives of Ophthalmology, New York, 1903, vol. xxxii. pp. 453–469.